A GLOBAL UPDATE OF LEAD POISONING IN TERRESTRIAL BIRDS FROM AMMUNITION SOURCES

DEBORAH J. PAIN[1], IAN J. FISHER[2], AND VERNON G. THOMAS[3]

[1]*Wildfowl and Wetlands Trust, Slimbridge, Gloucestershire, GL2 7BT, UK*
E-mail: debbie.pain@wwt.org.uk

[2]*Royal Society for the Protection of Birds, The Lodge, Sandy, Bedfordshire SG19 2DL, UK*

[3]*Department of Integrative Biology, College of Biological Science, University of Guelph, Guelph, ON N1G 2W1, Canada.*

ABSTRACT.—Lead poisoning mortality, through the ingestion of spent shot, is long established in waterfowl, and more recently in raptors and other avian taxa. Raptors (vultures, hawks, falcons, eagles and owls) are exposed to lead from spent ammunition (shot, bullets, or fragments from either) while feeding on game species, and other avian taxa are exposed when feeding in shot-over areas, including shooting ranges. Here we review the published literature on ingestion of and poisoning by lead from ammunition in terrestrial birds. We briefly discuss methods of evaluating exposure to and poisoning from ammunition sources of lead, and the use of lead isotopes for confirming the source of lead. Documented cases include 33 raptor species and 30 species from *Gruiformes*, *Galliformes* and various other avian taxa, including ten Globally Threatened or Near Threatened species. Lead poisoning is of particular conservation concern in long-lived slow breeding species, especially those with initially small populations such as the five Globally Threatened and one Near Threatened raptor species reported as poisoned by lead ammunition in the wild. Lead poisoning in raptors and other terrestrial species will not be eliminated until all lead gunshot and rifle bullets are replaced by non-toxic alternatives. *Received 29 May 2008, accepted 24 July 2008*.

PAIN, D. J., I. J. FISHER, AND V. G. THOMAS. 2009. A global update of lead poisoning in terrestrial birds from ammunition sources. *In* R. T. Watson, M. Fuller, M. Pokras, and W. G. Hunt (Eds.). Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans. The Peregrine Fund, Boise, Idaho, USA. DOI 10.4080/ilsa.2009.0108

Key words: Ammunition, global, lead, poisoning, terrestrial birds.

TERRESTRIAL BIRDS AT RISK

LEAD POISONING IN WILDFOWL AND WADERS from the ingestion of spent lead gunshot has been extensively studied, documented and reviewed over the last half century (Bellrose 1959, Kaiser and Fry 1980, Hall and Fisher 1985, Veiga 1985, Pain 1990a, 1990b, 1991a, 1992, 1996, Locke and Friend 1992, Sharley et al. 1992, Scheuhammer and Norris 1995, Beck and Granval 1997). Poisoning from the ingestion of lead from both gunshot and bullets by terrestrial birds initially received less attention, but today a considerable and increasing literature exists. While ingestion of discharged lead from firearms has been recorded in a diverse range of terrestrial birds, two groups of birds are particularly susceptible.

One group includes birds that are exposed in a similar way to wildfowl and waders, while feeding in any shot over areas where spent lead is deposited. Such areas include upland and lowland game shooting areas, shooting ranges including clay-pigeon shoots, and areas where species such as squirrels and other rodents are shot as pests. Exposure is likely to be higher in species that, like wildfowl, deliberately ingest grit to help break down food and/or ingest food items similar in appearance to lead shot (such as seeds) and may therefore mistakenly ingest lead. Many species fall into this category, but species are predominantly *Gruiformes* and *Galliformes* (Table 1). The second group at risk comprise species that ingest lead from bullets or shot while preying upon or scavenging game or other species of mammals or birds (such as 'pest' species, e.g., Knopper et al. 2006) that have been shot but survived, or killed but unretrieved. Species at risk are predominantly raptors, herein defined as vultures (New and Old World), hawks, falcons, eagles and owls (Table 1). Here, risk of exposure is likely to be particularly high in species that feed more frequently on hunter-shot quarry and have a propensity to scavenge. Species are also more likely to be exposed if there is intensive hunting within their foraging range (e.g. Mateo et al. 1998a, Wayland and Bollinger 1999) and during the hunting season (Elliott et al. 1992, Pain et al. 1997).

One of the earliest reports of lead poisoning from lead shot ingestion in a non-wildfowl species involved a Ring-necked Pheasant (*Phasianus colchicus*) in the UK (Calvert 1876), and a recent study from British shooting estates found an overall shot ingestion rate of 3%, with correspondingly elevated bone lead levels (Butler et al. 2005). Evidence for lead poisoning in this species therefore spans 125 years. Species of *Galliformes* and *Gruiformes* have been reported to have ingested shot and/or suffered lead poisoning through exposure to lead shot in a wide range of countries across Europe, and North America (Table 1).

In areas where lead ammunition is used, all raptors that feed on game or other hunted species are potentially at risk from lead poisoning. Species that take live prey, such as the Goshawk (*Accipiter gentilis*), are exposed through ingesting animals that have been shot but survived carrying shot in their flesh. In some cases exposure rates can be high, e.g., 25% and 36% of first year and adult live-trapped Pink-footed Geese (*Anser brachyrhynchus*) carried shot (Noer and Madsen 1996). However, species that scavenge, or both hunt and scavenge, are likely to be at particular risk from lead ingestion through feeding on injured or hunter-killed but unretrieved prey. Season and local hunting intensity play an important part in determining the level of dietary lead exposure (Pain et al. 1993, Mateo 1998a). For example, of Bald Eagles (*Haliaeetus leucocephalus*) found sick, injured or dead with significantly elevated lead exposure in British Columbia, the greatest number were found between January and March when they were feeding heavily on wintering waterfowl (Elliott et al. 1992). The prevalence of Bald Eagles with elevated lead exposure was found to be higher in areas of high waterfowl hunting intensity than areas with low hunting intensity (Wayland and Bollinger 1999). Similarly, Pain et al. (1997) found a significantly higher prevalence of elevated blood lead concentrations in Marsh Harriers (*Circus aeruginosus*) in France during than outside the hunting season.

Raptors are at risk both from prey killed with shotguns, such as wildfowl, rabbits and rodents, and with rifles, such as deer and other large game. The prevalence of exposure to lead from bullets is likely to be higher in larger raptors, and poisoning from bullet fragments has been reported in wild Bald Eagles, Golden Eagles (*Aquila chrysaetos*), Steller's Sea Eagles (*Haliaeetus pelagicus*), White-tailed Eagles (*H. albicilla*), and California Condors (*Gymnogyps californianus*) (Table 1).

Both captive and supplementary-fed wild birds may also be at risk if food includes animals that have been killed with lead ammunition, even when shot and bullets have been removed (e.g., Pain et al. 2007). Until recently, the extent to which lead shot and bullets fragment while passing through the tissues of prey was not realised. However, radiographs have shown that both shot and bullets undergo considerable fragmentation and fragments may be both distant from the wound canal, and too small to be visually detected (e.g., Pain et al. 2007). Many authors have now shown that feeding on species killed by lead gunshot or bullets can present a significant risk to scavengers, whether or not they

are exposed to whole shot or bullets (Scheuhammer et al. 1998, Sergeyev and Shulyatieva 2005, Hunt et al. 2006, Knopper et al. 2006, Pain et al. 2007). For example, Scheuhammer et al. (1998) found that 11% (92 of 827) of pectoral muscle pools from hunter-killed game (predominantly waterfowl) had elevated lead concentrations, in excess of 0.5 μg g^{-1} ww (mean of 12± 38 μg g^{-1}ww). While no lead could be detected visibly in the samples, radiography showed tiny embedded metal fragments.

EXPOSURE TO LEAD GUNSHOT AND BULLETS

Lead exposure is usually measured through the presence of lead shot or lead fragments in pellets regurgitated by raptors (e.g., Platt 1976, Pain et al. 1997, 2007), or the presence of shot, bullets or fragments in the alimentary tracts of birds post-mortem (e.g. Church et al. 2006 and numerous authors in Table 1).

The regurgitation of ingested shot in pellets illustrates that not all exposure to lead need be fatal, and regurgitation will undoubtedly reduce the amount of lead absorbed and the level of poisoning in raptors. However, repeated exposure and regurgitation of lead shot can result in poisoning (Pattee et al. 1981). In areas of high exposure, the likelihood of picking up shot (and of repeated exposure) increases. For example, as Bald Eagles and their waterfowl prey become concentrated in smaller areas, the likelihood of eagles picking up shot increases (Pattee and Hennes 1983). Consequently, exposure measured through the presence of shot in regurgitated pellets is likely to give a good indication of the likelihood of a species to become poisoned. Pain et al. (1997) found that the incidence of shot in regurgitated Marsh Harrier pellets dropped from up to 25% during the hunting season to just 1.4% after, in parallel with a significant reduction in blood lead concentrations in the birds outside the hunting season. Other studies have similarly found both the presence of lead shot in regurgitated pellets and evidence of poisoning and mortality from lead in wild birds (e.g., Red Kites [*Milvus milvus*] in the UK, Pain et al. 2007).

The presence of lead particles in the alimentary tracts of birds at post mortem is a widely used indicator of exposure, and both the number of lead shot or fragments found and the degree of abrasion or dissolution may provide supplementary evidence to help in the diagnosis of lead poisoning. Occasionally, large numbers of shot are ingested, and in Spain, where Golden Eagles have been found with lead toxicosis from shot ingestion, one bird had 40 pellets in its proventriculus (Cerradelo et al. 1992).

It is possible that birds that have not ingested lead from ammunition but carry shot-in lead in their tissues may also have elevated blood lead concentrations, as has been found in humans that have been shot with lead bullets (McQuirter et al. 2004), although these are likely to be far lower than in birds that have ingested lead fragments.

EVALUATING LEAD POISONING

Lead is not an essential element, and is a non-specific poison affecting all body systems. Lead poisoned birds often exhibit a distended proventriculus, green watery faeces, weight loss, anaemia and drooping posture, among other signs (Redig et al. 1980, Reiser and Temple 1981, Franson et al. 1983, Custer et al. 1984, Sanderson and Bellrose 1986, Friend 1987, Mateo 1998b). Lead affects the nervous system, kidneys and the circulatory system, resulting in a range of sub-lethal physiological, biochemical and behavioural changes (Scheuhammer 1987). There is no true 'no-effect' threshold level for lead; for example even at extremely low concentrations lead depresses the activity of the blood enzyme delta aminolevulinic acid, essential for haemoglobin production (Grasman and Scanlon 1995, Redig et al. 1991).

Once ingested, lead ammunition or fragments are readily dissolved in the acidic conditions in the intestine, and in birds that ingest grit to aid digestion (e.g., some *Gruiformes* and *Galliformes*) abrasion of the lead may accelerate this process. Directly following absorption the highest lead concentrations tend to be found in the blood, from which lead is transported around the body and deposited in soft tissue and bone. Blood lead generally has a half-life of around two weeks in birds (e.g., 14 days in the California Condor, Fry et al. 2009 this volume), whereas liver and kidney tissues generally retain elevated lead concentrations for weeks to several

Table 1. Lead shot ingestion and poisoning.

Species	Status	Evidence	Countries	References
Common Raven (*Corvus corax)*	LC	Poisoning	USA	Craighead and Bedrosian 2008
Chukar (*Alectoris chukar*)	LC	Ingestion	USA	Hanspeter and Kerry 2003
Grey Partridge (*Perdix perdix*)	LC	Ingestion and Poisoning	Denmark, UK	Clausen and Wolstrup 1979, Keymer and Stebbings 1987, Potts, 2005
Common Pheasant (*Phasianus colchicus)*	LC	Ingestion and Poisoning	Denmark, UK, USA	Calvert 1876, Elder 1955, Clausen and Wolstrup 1979, NWHL 1985, Dutton and Bolen 2000, Butler et al. 2005.
Wild Turkey (*Meleagris gallopavo*)	LC	Ingestion	USA	Stone and Butkas 1978
Scaled Quail (*Callipepla squamata*)	LC	Ingestion	USA	Campbell 1950
Northern Bobwhite Quail (*Colinus virginianus*)	NT	Ingestion	USA	Stoddard 1931, Keel et al. 2002
Great Horned Owl (*Bubo virginianus*)	LC	Poisoning	Canada	Clark and Scheuhammer 2003
Eurasian Eagle Owl (*B. bubo*)	LC	Poisoning	Spain	Mateo et al. 2003
Snowy Owl (*Nyctea scandiaca*)	LC	Poisoning	Captive	MacDonald et al. 1983
Long-eared Owl (*Asio otus*)	LC	Poisoning	Spain	Brinzal 1996
Rock Pigeon (*Columba livia*)	LC	Ingestion	USA , Belgium	Dement et al. 1987, Tavernier et al. 2004
Common Wood-pigeon (*C. palumbus*)	LC	Poisoning	Denmark	Clausen and Wolstrup 1979
Mourning Dove (*Zenaida macroura*)	LC	Ingestion	USA	Locke and Bagley 1967, Lewis and Legler 1968, Best et al. 1992, Schulz et al. 2002
Sandhill Crane (*Grus canadensis*)	LC	Ingestion	USA	Windingstad et al. 1984, NWHL 1985
Whooping Crane (*G. americana*)	EN	Poisoning	USA	Hall and Fisher 1985
Clapper Rail (*Rallus longirostris*)	LC	Ingestion	USA	Jones 1939
King Rail (*R. elegans*)	LC	Ingestion	USA	Jones 1939
Virginia Rail (*R. limicola*)	LC	Ingestion	USA	Jones 1939
Sora (*Porzana carolina*)	LC	Poisoning	USA	Jones 1939, Artman and Martin 1975, Stendell et al. 1980
Common Moorhen (*Gallinula chloropus*)	LC	Ingestion	Europe, USA	Jones 1939, Locke and Friend 1992
Common Coot (*Fulica atra*)	LC	Ingestion	France	Pain 1990a
American Coot (*F. americana*)	LC	Ingestion	USA	Jones 1939

Species	Status	Evidence	Countries	References
American Woodcock (*Scolopax minor*)	LC	Ingestion	Canada	Scheuhammer et al. 1999, 2003
Ruffed Grouse (*Bonasa umbellus*)		Ingestion	Canada	Rodrigue et al. 2005
California Gull (*Larus californicus*)	LC	Ingestion	USA	Quortrup and Shillinger 1941
Glaucous-winged Gull (*L. glaucescens*)	LC	Ingestion	USA	NWHL 1985
Herring Gull (*L. argentatus*)	LC	Ingestion	USA	NWHL 1985
European Honey-buzzard (*Pernis apivorus*)	LC	Unknown (ingestion or shot)	Netherlands	Lumeiji et al. 1985
Red Kite (*Milvus milvus*)	LC	Ingestion or poisoning	Germany, Spain, UK, Captive	Mateo 1998a, Mateo et al. 2001, 2003, Pain et al. 1997, Kenntner et al. 2005
Bald Eagle (*Haliaeetus leucocephalus*)	LC	Poisoning, shot and bullets	Canada, USA	Platt 1976, Jacobson et al. 1977, Kaiser et al. 1980, Pattee and Hennes 1983, Reichel et al. 1984, Frenzel and Anthony 1989, Craig et al. 1990, Langelier et al. 1991, Elliott et al. 1992, Gill and Langelier 1994, Scheuhammer and Norris 1996, Wayland and Bollinger 1999, Miller et al. 2000, 2001, Clark and Scheuhammer 2003, Wayland et al. 2003
Steller's Sea-eagle (*H. pelagicus*)	VU	Poisoning, bullets	Japan	Kim et al. 1999, Iwata et al. 2000, Kurosawa 2000
White-rumped Vulture (*Gyps bengalensis*)	CR	Poisoning (origin unknown)	Pakistan	Oaks et al. 2004
Eurasian Griffon (*G. fulvus*)	LC	Poisoning	Spain	Mateo et al. 1997, Guitart 1998, Mateo et al. 2003, Garcia-Fernandez et al. 2005
Egyptian Vulture (*Neophron percnopterus)*	EN	Poisoning	Canary Islands; Iberian peninsula	Donazar et al. 2002, Rodriguez-Ramos et al. 2009, this volume
Cinereous Vulture *(Aegypius monachus)*	NT	Poisoning	Spain	Hernandez and Margalida 2008
Eastern Marsh-harrier (*Circus spilonotus)*		Ingestion	Japan	Hirano et al. 2004
Western Marsh-harrier (*Circus aeruginosus*)	LC	Poisoning	France, Germany, Spain	Pain et al. 1993, 1997, Mateo et al., 1999, Kenntner et al. 2005
Northern Harrier (*C. cyaneus*)	LC	Ingestion	Canada, USA	Martin and Barrett 2001, Martin et al. 2003
Eurasian Sparrowhawk (*Accipiter nisus*)	LC	Ingestion	France, Captive	MacDonald et al. 1983, Pain and Amiard-Triquet 1993
Sharp-shinned Hawk (*A. striatus*)	LC	Ingestion	Canada, USA	Martin and Barrett 2001
Cooper's Hawk (*A. cooperii*)	LC	Ingestion	Canada, USA	Snyder et al. 1973, Martin and Barrett 2001
Northern Goshawk (*A. gentilis*)	LC	Poisoning	Canada, France, Germany, USA, Captive	Stehle 1980, Pain and Amiard-Triquet 1993, Martin and Barrett 2001, Kenntner et al. 2003, 2005

Species	Status	Evidence	Countries	References
Red-tailed Hawk (*Buteo jamaicensis*)	LC	Poisoning	Canada, USA	Franson et al. 1996, Martin and Barrett 2001, Clark and Scheuhammer 2003, Martina et al. 2008
Common Buzzard (*B. buteo*)	LC	Poisoning	France, Germany; UK, Italy, Captive	Stehle 1980, MacDonald et al. 1983, Pain and Amiard-Triquet 1993, Pain et al. 1995, Kenntner et al. 2005, Battaglia et al. 2005
Rough-legged Buzzard (*B. lagopus*)	LC	Poisoning	USA	Locke and Friend 1992
Spanish Imperial Eagle (*Aquila adalberti*)	VU	Poisoning	Spain	González and Hiraldo 1988, Hernández 1995, Mateo 1998a, Mateo et al. 2001, Pain et al. 2004
Golden Eagle (*A. chrysaetos*)	LC	Poisoning	Canada, Germany, Spain, Switzerland, USA	Bloom et al. 1989, Craig et al. 1990, Pattee et al. 1990, Cerradelo et al. 1992, Bezzel and Fünfstück 1995, Scheuhammer and Norris 1996, Wayland and Bollinger 1999, Clark and Scheuhammer 2003, Wayland et al. 2003, Kenntner et al. 2007
White-tailed Eagle (*Haliaeetus albicilla*)	LC	Ingestion and Poisoning – shot and bullets	Greenland, Poland; Austria, Germany, Japan, Finland	Falandysz et al. 1988, Kim et al. 1999, Iwata et al. 2000, Kurosawa 2000, Kenntner et al. 2001, 2004, 2005, Krone et al. 2004, 2006, Kalisinska et al. 2006
American Kestrel (*Falco sparverius*)	LC	Ingestion	Canada, USA	Martin and Barrett 2001
Laggar Falcon (*F. jugger*)	NT	Poisoning	Captive	MacDonald et al. 1983
Prairie Falcon (*F. mexicanus*)	LC	Poisoning	Captive	Benson et al. 1974, Stehle 1980
Peregrine Falcon (*F. peregrinus*)	LC	Poisoning	UK, Captive	MacDonald et al. 1983, Pain et al. 1995
Turkey Vulture (*Cathartes aura*)	LC	Ingestion	Canada, USA	Wiemeyer et al. 1986, Martin et al. 2003, Martina et al. 2008
California Condor (*Gymnogyps californianus*)	CR	Poisoning, bullets	USA	Wiemeyer et al. 1983, 1986, 1988, Janssen et al. 1986, Bloom et al. 1989, Pattee et al. 1990, Meretsky et al. 2000, Snyder and Snyder 2000
Andean Condor (*Vultur gryphus*)	NT	Poisoning	Captive	Locke et al. 1969
King Vulture (*Sarcorhampus papa*)	LC	Poisoning	Captive	Decker et al. 1979
White-throated Sparrow (*Zonotrichia albicollis*)	LC	Ingestion	USA	Vyas et al. 2000
Dark-eyed Junco (*Junco hyemalis*)	LC	Ingestion	USA	Vyas et al. 2000
Brown-headed Cowbird (*Molothrus atar*)	LC	Ingestion	USA	Vyas et al. 2000
Yellow-rumped Warbler (*Dendroica coronata*)		Poisoning	USA	Lewis et al. 2001
Brown Thrasher (*Toxostoma rufum*)		Poisoning	USA	Lewis et al. 2001
Solitary Vireo (*Vireo solitarius*)		Poisoning	USA	Lewis et al. 2001

Countries are given for wild birds; captive birds are simply listed as captive, and were poisoned through shot present in their feed. Conservation status is from BirdLife International (2004) and is coded as: CR—Critically Endangered, EN—Endangered , NT—Near Threatened, LC—Least Concern. Evidence of poisoning indicates tissue lead concentrations indicative of poisoning (e.g., Franson 1996) with the source of poisoning most likely to be lead gunshot and/or diagnosis of lead poisoning in one or more individuals; 'Ingestion' indicates evidence of ingestion of shot usually in the absence of tissue analysis. Rare cases of lead poisoning believed to be from non-ammunition sources are excluded.

Table 2. Guidelines for interpretation of tissue lead concentrations in *Falconiformes* and *Galliformes* (ppm ww).

	Falconiformes (ppm ww)			Galliformes (ppm ww)		
	Blood	**Liver**	**Kidney**	**Blood**	**Liver**	**Kidney**
Sub clinical	0.2–1.5	2–4	2–5	0.2–3	2–6	2–20
Toxic	>1	>3	>3	>5	>6	>15
Compatible with death	>5	>5	>5	>10	>15	>50

Adapted from Franson (1996)

months following absorption. Once deposited in bone, lead is far less mobile, and bone lead concentrations tend to remain elevated for months to years, reflecting lifetime exposure (Pain 1996). Several authors have suggested guidelines to help interpret tissue lead concentrations in different avian taxa (Franson 1996, Pain 1996, Table 2). Differences in sensitivity exist both among and within taxa (e.g., Table 2, Carpenter et al. 2003), and other factors, such as the duration and level of exposure, may influence the tissue lead concentrations at which effects are observed. There is no simple way of relating tissue lead concentrations to effect. However, the general conclusions on tissue lead levels associated with sub-lethal poisoning, toxicity, and death from lead poisoning that have been drawn from the vast field and experimental data reported in the published literature are useful guidelines (Table 2, Franson 1996).

The definition of 'background' lead concentrations is rather more complex and will differ with the context of the question being asked and the population under study. Natural levels of exposure to lead in the environment no longer exist as lead resulting from anthropogenic emissions is ubiquitous. Concentrations in the environment distant from emission sources are generally described as 'background.' In remote areas most birds would be expected to have <2 and certainly <5 µg/dL blood lead from exposure to background levels. However, populations of scavengers that feed in urban areas or at rubbish dumps may have median population blood lead concentrations far higher than this as they will be exposed to a mix of anthropogenic sources of lead. These higher concentrations could still usefully be defined as 'background for this population' when attempting to distinguish between low level chronic exposure from a mix of general environmental sources, and acute exposure, for example through the ingestion of lead from ammunition.

The diagnosis of lead poisoning is usually based upon clinical signs of poisoning in combination with blood lead concentrations in live birds. In dead birds, diagnosis is based upon tissue (liver or kidney) lead concentrations and clinical signs of poisoning, sometimes, but not necessarily, in combination with evidence of exposure to lead. Clinical signs of poisoning and/or evidence of exposure alone are insufficient to positively diagnose lead poisoning and tissue lead analysis is required. The presence of ingested lead objects in the intestine, while a good indicator, is not diagnostic of poisoning as little absorption may have occurred if objects have been ingested very recently. Similarly, the absence of lead objects in the intestine does not mean that birds have not been lead poisoned, as lead objects may be regurgitated or passed through after considerable absorption has occurred, or may be ground down and/or totally dissolved and absorbed.

Clinical signs are more frequently recorded in cases of chronic poisoning, whereas acutely poisoned birds may die rapidly in apparently good body condition (e.g., Gill and Langelier 1994). Liver and kidney lead concentrations are most useful in lead poisoning diagnosis post-mortem. Bone lead concentrations are less useful as, in cases of acute poisoning, lead poisoned birds may die rapidly with comparatively low bone lead concentrations, and conversely, high bone lead concentrations may result from chronic lifetime exposure in the absence of lead toxicity. Except in cases of acute exposure, bone lead concentrations have been shown to be positively correlated with age in waterfowl populations (Stendell et al. 1979, Clausen et al. 1982).

IDENTIFYING THE SOURCE OF LEAD

There are relatively few sources of lead to which exposure is sufficient to result in tissue concentrations indicative of toxicity (Table 2) or to result in mortality. By far the majority of cases in birds result from the ingestion of shot or bullet fragments (Table 1), or anglers' lead weights in certain water birds (Scheuhammer and Norris 1995). Rare cases result from point source exposure, e.g., to mine waste (Henny et al. 1991, 1994, Sileo et al. 2001) or industrial pollution (Bull et al. 1983), or the ingestion of lead in paint (Sileo and Fefer 1987). In cases where lead poisoning has been diagnosed and lead shot and/or bullet fragments are found in the bird's intestine, the likely source is self-evident. In most cases where no lead from ammunition is present, knowledge of feeding habits or areas of the species is often sufficient to conclude that lead from ammunition is the likely source.

One additional tool to help identify likely sources of lead contamination is the examination of the distribution of blood or soft tissue lead concentrations in a population of birds. Blood and soft tissue lead concentrations in birds that have simply been exposed to generally distributed anthropogenic lead (e.g., from atmospheric deposition) should follow a relatively normal distribution. Dramatic outliers from this, or distributions particularly skewed to higher lead concentrations, suggest elevated lead ingestion from an additional source in a proportion of birds. Distributions are likely to be normal with a few elevated outliers in birds that infrequently ingest lead ammunition, and heavily skewed to elevated concentrations, with more very elevated outliers in populations that more frequently ingest lead ammunition. Although the examination of population lead distributions is a useful indicator and can provide supporting evidence, and while lead from ammunition is by far the most frequently reported source of exposure, most distributions could potentially result from other forms of exposure, and the examination of tissue lead distributions should simply be used as a tool.

Recently, lead isotopes have been used to help source lead exposure (e.g., Meharg et al. 2002). Ratios of lead isotopes, $Pb^{206, 207, 208}$, can vary with the origin of the lead. For example, lead in background sources such as soil and air may have markedly different isotopic ratios from certain industrial sources, or from lead mined in different geographical regions. The utility of lead isotopes for distinguishing sources of lead contamination in wild birds was illustrated by Scheuhammer and Templeton (1998). They compared isotopic ratios in lead-exposed waterfowl and eagles and found them to be similar to those from lead shot pellets, whereas juvenile Herring Gulls (*Larus argentatus*) had isotopic ratios within the range characterising lead from gasoline combustion. Stable lead isotope ratios were used by Martin and Barrett (2001) to determine the source of lead exposure to wildlife on the north shore of Lake Erie. While none of the migrating birds sampled had lead levels indicating lead poisoning, one or more individuals of the following species were found to have levels indicative of sub-lethal lead exposure, with the lead isotope ratios for most of the samples falling within the range of gunshot pellets: American Kestrel (*Falco sparverius*), Sharp-shinned Hawk (*Accipiter striatus*), Cooper's Hawk, Northern Goshawk, Northern Harrier (*Circus cyaneus*), and Red-tailed Hawk (*Buteo jamaicensis*). More recently, Pain et al. (2007) found lead isotope ratios in the livers of Red Kites in the UK to be consistent with ratios in lead pellets, and Church et al. (2006) found that free-flying California Condors with relatively high blood lead concentrations had isotope values consistent with lead from ammunition and distinct from those of pre-release condors with low blood lead concentrations.

In most cases of poisoning from lead ammunition sources, there is sufficient knowledge of the feeding habits of birds, and their proclivity to ingest lead shot and bullet fragments to negate the need for isotopic studies. Isotopic studies may be particularly useful in those rare cases when the source of lead is unclear or where unusual patterns of tissue lead concentrations are found.

CASE STUDIES

Many lead poisoning studies are based upon knowledge of a species' feeding habits and ranges, the likelihood of shot and bullet fragment ingestion, and tissue lead concentrations, or upon the presence of ingested shot or bullet fragments. While these

factors are generally sufficient to evaluate lead poisoning from ammunition sources, many other studies have included a much wider range of data. A few illustrative examples are outlined here. Evidence for lead poisoning of Marsh Harriers in southern Europe came from (1) knowledge of the propensity of Marsh Harriers to scavenge waterfowl, (2) temporal variation in the proportion of regurgitated pellets containing lead shot, with far higher incidence during the hunting season and, (3) temporal variation in the proportion of birds with elevated blood lead concentrations, with a far higher incidence during than outside the hunting season (Pain et al. 1993, 1997, Mateo et al. 1999). In the UK, evidence for lead shot poisoning of Red Kites included (1) knowledge of propensity to scavenge game and other shot species, (2) the presence of lead gunshot in regurgitated pellets, (3) tissue lead concentrations consistent with lead poisoning mortality at post mortem, and (4) lead isotope values in the tissues of dead birds with elevated lead concentrations consistent with those of lead shot (Pain et al. 1997). By far the most comprehensively studied species is the California Condor, for which evidence for lead poisoning from spent shot and bullets includes all of the above. It includes: post-mortem evidence and/or evidence of ammunition ingestion, moribund birds with clinical signs of lead poisoning and elevated blood lead levels, elevated blood lead levels in free-flying birds, temporal and spatial correlations between big game hunting seasons and areas and elevated lead levels in condors, and lead isotope ratios in the tissues of exposed condors consistent with those of lead shot and bullets (Church et al. 2006 and authors in Table 1). This provides only a small sample of the numerous studies that combine a wide range of evidence of lead poisoning from ammunition sources in individual species.

SPECIES AFFECTED BY LEAD POISONING

Cases of lead shot ingestion and/or poisoning in terrestrial birds are documented in Table 1. This currently includes 33 raptor species and 30 species from *Gruiformes, Galliformes* and various other avian taxa. Twenty-eight raptor species and all species from other taxa were free-flying, and an additional ten raptor species were poisoned accidentally in captivity from lead shot in food carcasses. This was usually hunter-killed rabbits, squirrels and other small mammals, including an example where shot were manually removed from prey, but where small lead fragments likely remained (Locke et al. 1969, Benson et al. 1974, Decker et al. 1979, Jacobsen et al. 1977, Stehle 1980, MacDonald et al. 1983, Pain et al. 1997). While most cases of poisoning are in raptors, *Gruiformes* or *Galliformes*, several species from other taxa have been shown to have ingested shot, including pigeons, doves, gulls, and a range of passerines.

In addition to published cases reviewed for this study, records of many other species poisoned by lead ammunition exist at veterinary schools and similar institutions (M. Pokras pers. comm.).

PHYSIOLOGICAL AND POPULATION IMPACTS OF LEAD POISONING

Lead poisoning through the ingestion of shot and bullet fragments by terrestrial birds causes a wide range of sub-lethal impacts affecting physiology and behaviour, and when exposure is sufficiently high or of sufficient duration results in mortality. Experimental studies in birds have shown that, among other effects, lead can impair blood synthesis, immune function and reproduction (Grandjean 1976, Kendall et al. 1981, Kendall and Scanlon 1981, Veit et al. 1982, Edens and Garlich 1983, Scheuhammer 1987, Grasman and Scanlon 1995, Redig et al. 1991). Consequently, free-living birds exhibiting sub-lethal poisoning will likely be more susceptible to disease, starvation and predation, and an increased probability of death from other causes (Scheuhammer and Norris 1996). Although the extent to which survival is reduced by sub-lethal lead poisoning is difficult to quantify in field studies, it is clearly undesirable in any species.

Mortality from lead poisoning is of greatest concern in long-lived, slow-breeding species, characteristics typical of many raptors, and particularly species with populations that are either naturally small or have been reduced by other factors. Such species are particularly vulnerable to increases in adult mortality, and all age classes can be affected by lead poisoning.

Lead poisoning in the Bald Eagle, a species whose population was depressed through losses due to pesticide abuses in the 1950s and 1960s, is well documented (Elliott et al. 1992, Pattee and Hennes 1983, Wayland and Bollinger 1999, Table 1). In Canada and the USA, approximately 10–15% of recorded post-fledging mortality in Bald and Golden Eagles was attributed to the ingestion of lead shot from prey animals (Scheuhammer and Norris 1996). Elliott et al. (1992) found that 14% of 294 sick, injured. or dead Bald Eagles in British Columbia (1988 to 1991) were lead-poisoned and an additional 23% sub-clinically exposed, with the majority of exposure occurring during January to March, when the birds were feeding heavily on wintering wildfowl.

Of particular concern is lead poisoning in globally threatened species. To date, eight globally threatened (Critically Endangered, Endangered or Vulnerable) or Near Threatened (BirdLife International 2008) free-living species have been reported as lead-poisoned (Table 1), of which six are raptors. While there were isolated reports in two cases, the Endangered Whooping Crane (*Grus americana*) and Critically Endangered Oriental White-backed Vulture (*Gyps bengalensis*), multiple reports exist for the other species. In addition, two captive Near Threatened species have been reported with lead poisoning from ammunition sources: Laggar Falcon (*Falco jugger*) and Andean Condor (*Vultur gryphus*). High levels of adult mortality occurred in globally Vulnerable Steller's Sea-eagles and White-tailed Eagles (Least Concern) in Hokkaido, Japan, until a ban on the use of lead bullets in this area (Kurosawa 2000). Population modelling indicated that in the absence of preventative measures Steller's Sea Eagle populations would decline as a result of lead poisoning, and that populations were most sensitive to changes in adult mortality (Ueta and Masterov 2000). The Vulnerable Spanish Imperial Eagle (*Aquila adelberti*) is an example of a species with a small population, estimated at 200 pairs (BirdLife International 2008) that cannot sustain high, especially adult, mortality (Ferrer 2001, Ferrer et al. 2003). The most significant impacts of lead poisoning on any terrestrial species have been on the Critically Endangered California Condor. Lead poisoning from lead shot and bullet fragments was a major factor in the decline of this species, which became extinct in the wild, and has seriously hampered reintroduction efforts (Meretsky et al. 2000, Cade 2007).

In addition to lead poisoning in globally threatened species, many of the species in Table 1 are of national or regional conservation concern (e.g., Gregory et al. 2002 for the UK).

Not only does lead poisoning cause unnecessary mortality in many terrestrial avian species, and present a significant problem at a population level to a few, it also severely compromises the welfare of large numbers of wild birds, both terrestrial and aquatic. Sainsbury et al. (1995) investigated the scale and severity of welfare issues in wild European birds and mammals. They described the nature and level of harm caused (pain, stress and fear), the duration of effects and numbers of individuals affected, and found lead poisoning through shot ingestion to be among the most significant human activities that severely compromise the welfare of large numbers of animals.

REMOVING THE THREAT OF LEAD POISONING IN TERRESTRIAL BIRDS

Lead poisoning from ammunition affects the populations of several globally threatened species, and hampers the reintroduction of the critically endangered California Condor. In many species it causes mortality or sub-lethal effects which may influence survival. Limited legislative steps have been taken to reduce the threat of lead poisoning to terrestrial birds, including nationwide legislation banning the use of lead shot for all waterfowl hunting in the USA from 1991, aimed at preventing secondary lead toxicosis of the Bald Eagle (Anderson 1992), and a ban on the use of lead bullets for hunting on Hokkaido, Japan, where secondary poisoning affected many Steller's Sea Eagles (Matsuda 2003). More recently, and in the face of considerable opposition from the gun lobby, the State of California passed Assembly Bill 821 in 2007 to restrict the use of lead shot and bullets in the range of the California Condor. Such legislation is an important step towards reducing the risk of lead poisoning in terrestrial birds; policing and compliance are also essential, and much remains to be done in these areas.

The risk to terrestrial and water birds, of lead poisoning from ammunition is just one of many reasons for replacing lead in all ammunition with non-toxic alternatives. Other reasons have been covered elsewhere in this volume, and include the following:

- Lead is a highly toxic heavy metal with no known biological function that affects humans and other animals at the lowest measurable concentrations. Lead has already been banned from most uses that could result in human and wildlife exposure (e.g., from paint, and as an anti-knocking agent and octane booster in gasoline/petrol).
- The use of lead ammunition results in huge quantities of lead deposited in the environment annually (Thomas 1997), where it is extremely persistent in contaminating soils and waterways (e.g., Sorvari et al. 2006) and augmenting potential future risks to wildlife inhabiting shot-over areas.
- Environmental clean-up following the deposition of lead ammunition is both difficult and costly.
- Some lead from ammunition sources is or becomes bioavailable and may be bioaccumulated by edaphic organisms (Migliorini et al. 2004), and puts at risk vegetation, invertebrates and other organisms (Bennett et al. 2007).
- Lead poisoning may severely compromise the welfare of large numbers of wild animals.
- Lead from ammunition can present a human health risk, e.g., at firing ranges and to people who consume game shot with lead (Scheuhammer et al. 1998, Tsuji et al. 1999, Guitart et al. 2002, Johansen et al. 2004, Gustavsson and Gerhardsson 2005). While not all people that consume game will necessarily develop elevated blood lead concentrations (e.g., Haldimann et al. 2002), those that frequently consume game such as subsistence hunters appear particularly at risk (e.g., Bjerregaard et al. 2004) as may be people who cook game using recipes of low pH that facilitate lead dissolution (e.g., Mateo et al. 2007).

There are few reasons to continue with the use of lead ammunition. Non-toxic shot regulations have been shown to be successful at reducing the incidence of lead poisoning (e.g., Anderson et al. 2000, Stevenson et al. 2005) and are unlikely to result in increased crippling rates (Schultz et al. 2006). Cade (2007) reviewed the compelling evidence for the impacts of lead poisoning from ammunition on the California Condor population, and concluded that the use of non-toxic shot and bullets would be highly efficacious for hunting, economically feasible, and ethically the right thing to do.

While previous regulatory emphasis has been placed on wetland habitats or waterfowl hunting, there is now an urgent and compelling need to replace all lead gunshot and rifle bullets for shooting game, pest species, and targets with non-toxic alternatives. The wide extent of primary and secondary lead poisoning has been demonstrated across a large range of bird species and continents wherein hunting has been practiced. The burden of proof that lead from spent gunshot and bullets causes lead toxicosis has already been achieved for wetland species, upland species, and avian predators and scavengers. More recently, the risks to humans have been documented, especially those who regularly eat hunted game meat (see the collection of papers in this volume). Thomas and Guitart (2005) indicated that several global treaties and agreements exist that could authorize the transition to non-toxic shot and bullets in many countries, and presage the passing of binding legislation to support bans on the use of lead products. The removal of lead shot, bullets and fishing weights from sporting use is also completely consistent with the Precautionary Principle (Matsuda 2003), and does not appear compromised by considerations of economic costs, commercial availability, or ballistic effectiveness. The transition to use of non-toxic lead substitutes is vital to ensure the sustainable use of wildlife, and to reduce the risks of lead poisoning from lead bullets and shot to human health.

LITERATURE CITED

ANDERSON, W. L. 1992. Legislation and lawsuits in the United States and their effects on non-toxic shot regulations. Pages 56–60 *in* D.J. Pain (Ed.). Lead Poisoning in Waterfowl. Proceedings of an IWRB Workshop, Brussels, Belgium, 13–15 June 1991. International Waterfowl and Wetlands Research Bureau Special Publication 16, Slimbridge, UK.

ANDERSON, W. L., S. P. HAVERA, AND B. W. ZERCHER. 2000. Ingestion of lead and non-toxic shotgun pellets by ducks in the Mississippi flyway. Journal of Wildlife Management 64:848–857.

ARTMANN, J. W., AND E. M. MARTIN. 1975. Incidence of ingested lead shot in Sora Rails. Journal of Wildlife Management 39:514–519.

BATTAGLIA, A., S. GHIDINI, G. CAMPANINI, AND R. SPAGGIARI. 2005. Heavy metal contamination in Little Owl (*Athene noctua*) and Common Buzzard (*Buteo buteo*) from northern Italy. Ecotoxicology and Environmental Safety 60:61–66.

BECK, N., AND P. GRANVAL. 1997. Ingestion de plombs de chasse par la Bécassine del Marais (*Gallinago gallinago*) et la Bécassine Sourde (*Lymnocryptes minimus*) dans le nord-ouest de la France. Gibier Faune Sauvage 14:65–70.

BELLROSE, F. C. 1959. Lead poisoning as a mortality factor in waterfowl populations. Illinois Natural History Survey Bulletin 27:235–288.

BENSON, W. W., B. PHARAON, AND P. MILLER. 1974. Lead poisoning in a bird of prey. Bulletin of Environmental Contamination Toxicology 11:105–108.

BENNETT, J. R., C. A. KAUFMAN, I. KOCH, J. SOVA, AND K. J. REIMER. 2007. Ecological risk assessment of lead contamination at rifle and pistol ranges using techniques to account for site characteristics. Science of the Total Environment 374:91–101.

BEST, T. L., T. E. GARRISON, AND C. G. SCHMITT. 1992. Availability and ingestion of lead shot by Mourning Doves (*Zenaida macroura*) in Southeastern New Mexico. The South-eastern Naturalist 37:287–292.

BEZZEL, E., AND H. J. FÜNFSTÜCK. 1995. Alpine Steinadler (*Aquila chrysaetos*) druch Bleivergiftung gefährdet? Journal für Ornithologie 136: 294–296.

BIRDLIFE INTERNATIONAL. 2008. Species factsheets downloaded from http://www.birdlife.org on 29 May 2008.

BJERREGAARD, P., P. JOHANSEN, G. MULVAD, H. S. PEDERSEN, AND J. C. HANSEN. 2004. Lead sources in human diet in Greenland. Environmental Health Perspectives 112:1496–1498.

BLOOM, P. H., J. M. SCOTT, O. H. PATTEE, AND M. R. SMITH. 1989. Lead contamination of Golden Eagles (*Aquila chrysaetos*) within the range of the Californian Condor (*Gymnogyps californianus*). Pages 481–482 *in* B-U. Meyburgh and R.D. Chancellor (Eds.). Raptors in the Modern World. Proceedings of the 3rd World Conference on Birds of Prey and Owls, Eliat, Israel, 22–27 March 1987. World Working Group on Birds of Prey, Berlin, London, and Paris.

BRINZAL. 1996. S.O.S. venenos: Búho Chico. Quercus 124:45.

BULL, K. R., W. J. EVERY, P. FREESTONE, J. R. HALL, AND D. OSBORN. 1983. Alkyl lead pollution and bird mortalities on the Mersey estuary, UK 1979–1981. Environmental Pollution 31: 239–259.

BUTLER, D. A., R. B. SAGE, R. A. H. DRAYCOTT, J. P. CARROLL, AND D. POTTS. 2005. Lead exposure in Ring-Necked Pheasants on shooting estates in Great Britain. Wildlife Society Bulletin 33: 583–589.

CADE, T. J. 2007. Exposure of California Condors to lead from spent ammunition. Journal of Wildlife Management 71:2125–2133.

CALVERT, J. H. 1876. Pheasants poisoned by swallowing shots. The Field 47:189.

CAMPBELL, H. 1950. Quail picking up lead shot. Journal of Wildlife Management 14:243–244.

CARPENTER, J. W., O. H. PATTEE, S. H. FRITTS, B. A. RATTNER, S. N. WIEMEYER, J. A. ROYLE, AND M. R. SMITH. 2003. Experimental lead poisoning in Turkey Vultures (*Cathartes aura*). Journal of Wildlife Diseases 39:96–104.

CERRADELO, S., E. MUÑOZ, J. TO-FIGUERAS, R. MATEO, AND R. GUITART. 1992. Intoxicación por ingestión de perdigones de plomo en dos Águilas Reales. Doñana, Acta Vertebrata 19: 122–127.

CHURCH, M. E., R. GWIAZDA, R. W. RISEBOROUGH, K. SORENSON, C. P. CHAMBERLAIN, S. FARRY, W. HEINRICH, B. A. RIDEOUT, AND D. R. SMITH. 2006. Ammunition is the principal source of lead accumulated by California Condors reintroduced to the wild. Environmental Science and Technology 40: 6143–6150.

CLARK, A. J., AND A. M. SCHEUHAMMER. 2003. Lead poisoning in upland-foraging birds of prey in Canada. Ecotoxicology 12:23–30.

CLAUSEN, B., AND C. WOLSTRUP. 1979. Lead poisoning in game from Denmark. Danish Review of Game Biology 11:22.

CLAUSEN, B., K. ELVESTAD, AND O. KARLOG. 1982. Lead burden in Mute Swans from Denmark. Nordisk Veterinaer Medicine 34:83–91.

CRAIG, T. H., J. W. CONNELLY, E. H. CRAIG, AND T. L. PARKER. 1990. Lead concentrations in Golden and Bald Eagles. Wilson Bulletin 102: 130–133.

CRAIGHEAD, D., AND B. BEDROSIAN. 2008. Blood lead levels of Common Ravens with access to big-game offal. Journal of Wildlife Management 72:240–245.

CUSTER, T. W., J. C. FRANSON, AND O. H. PATTEE. 1984. Tissue lead distribution and hematologic effects in American Kestrels (*Falco sparverius*) fed biologically incorporated lead. Journal of Wildlife Diseases 20: 39–43.

DECKER, R. A., A. M. MCDERMID, AND J. W. PRIDEAUX. 1979. Lead poisoning in two captive King Vultures. Journal of the American Medical Association 175:1009.

DEMENT, S. H., J. J. CHISOLM, JR., M. A. ECKHAUS, AND J. D. STRANDBERG. 1987. Toxic lead exposure in the urban Rock Dove. Journal of Wildlife Diseases 23:273–278.

DONAZAR, J. A., C. J. PALACIOS, L. GANGOSO, O. CEBALLOS, M. J. GONZALEZ, AND F. HIRALDO. 2002. Conservation status and limiting factors in the endangered population of Egyptian Vulture (*Neophron percnopterus*) in the Canary Islands. Biological Conservation 107:89–97.

DUTTON, C. S., AND E. G. BOLEN. 2000. Fall diet of a relict pheasant population in North Carolina. Journal of the Elisha Mitchell Scientific Society 116:41–48.

EDENS, F. W., AND J. D. GARLICH. 1983. Lead-induced egg production decrease in leghorn and Japanese quail hens. Poultry Science 64:1757–1763.

EISLER, R. 1988. Lead hazards to fish, wildlife, and invertebrates: a synoptic review. United States Fish and Wildlife Service Biological Report 85: 1–4.

ELDER, W. H. 1955. Fluoroscope measures of hunting pressure in Europe and North America. Transactions of the North American Wildlife Conference 20: 298–322.

ELLIOTT, J. E., K. M. LANGELIER, A. M. SCHEUHAMMER, P. H. SINCLAIR, AND P. E. WHITEHEAD. 1992. Incidence of lead poisoning in Bald Eagles and lead shot in waterfowl gizzards from British Columbia 1988–91. Canadian Wildlife Service Program Note No. 200. Canadian Wildlife Service, Quebec, Canada.

FALANDYSZ, J., B. JAKUCZUN, AND T. MIZERA. 1988. Metal and organochlorines in four female White-tailed Eagles. Marine Pollution 19:521–526.

FERRER, M. 2001. The Spanish Imperial Eagle. Lynx Editions, Barcelona, Spain.

FERRER, M., V. PENTERIANI, J. BALBONTÍN, AND M. PANDOLFI. 2003. The proportion of immature breeders as a reliable early warning signal of population decline: evidence from the Spanish Imperial Eagle in Doñana. Biological Conservation 114:463–466.

FRANSON, J. C. 1996. Interpretation of tissue lead residues in birds other than waterfowl. Pages 265–279 *in* W. N. Beyer, G. H. Heinz, and A. W. Redmon-Norwood (Eds.). Environmental Contaminants in Wildlife: Interpreting Tissue Concentrations. SETAC, CRC/Lewis Publishers, Boca Raton, Florida, USA.

FRANSON, J. C., L. SILEO, O. H. PATTEE, AND J. F. MOORE. 1983. Effects of chronic dietary lead in American Kestrels (*Falco spaverius*). Journal of Wildlife Diseases 19:110–113.

FRANSON, J. C., N. J. THOMAS, M. R. SMITH, A. H. ROBBINS, S. NEWMAN, AND P. C. MCCARTIN. 1996. A retrospective study of post-mortem findings in Red-tailed Hawks. Journal of Raptor Research 30: 7–14.

FRENZEL, R. W., AND R. G. ANTHONY. 1989. Relationship of diets and environmental contaminants in wintering Bald Eagles. Journal of Wildlife Management 53:792–802.

FRIEND, M. 1987. Field guide to wildlife diseases. United States Fish and Wildlife Service, Washington, DC, USA.

FRY, M., K. SORENSON, J. GRANTHAM, J. BURNETT, J. BRANDT, AND M. KOENIG. 2009. Lead Intoxication Kinetics in Condors from California. Abstract *in* R. T. Watson, M. Fuller, M. Pokras, and W. G. Hunt (Eds.). Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans. The Peregrine Fund, Boise, Idaho, USA. DOI 10.4080/ilsa.2009.0301

GARCIA-FERNANDEZ, A. J., E. MARTINEZ-LOPEZ, D. ROMERO, P. MARIA-MOJICA, A. GODINO, AND P. JIMENEZ. 2005. High levels of blood

lead in Griffon Vultures (*Gyps fulvus*) from Cazorla Natural Park (southern Spain). Environmental Toxicology 20: 459–463.

GILL, E., AND K. M. LANGELIER. 1994. Acute lead poisoning in a Bald Eagle secondary to bullet ingestion. Canadian Veterinary Journal 35:303–304.

GONZÁLEZ, L. M., AND F. HIRALDO. 1988. Organochlorine and heavy metals contamination in the eggs of the Spanish Imperial Eagle (*Aquila adalberti*) and accompanying changes in eggshell morphology and chemistry. Environmental Pollution 51:241–258.

GRANDJEAN, P. 1976. Possible effect of lead on eggshell thickness in kestrels 1874–1974. Bulletin of Environmental Contamination and Toxicology 16:101–106.

GRASMAN, K. A. AND P. F. SCANLON. 1995. Effects of acute lead ingestion and diet on antibody and T-cell-mediated immunity in Japanese quail. Archives of Environmental Contamination and Toxicology 28:161–167.

GREGORY, R. D., N. I. WILKINSON, D. G. NOBLE, J. A. ROBINSON, A. F. BROWN, J. HUGHES, D. PROCTER, D. W. GIBBONS, AND C. GALBRAITH. 2002. The population status of birds in the United Kingdom, Channel Islands and Isle of Man: an analysis of conservation concern 2002–2007. British Birds 95:410–48.

GUITART, R. 1998. Plumbismo en buitres. Quercus 146:47.

GUITART, R., J. SERRATOSA, AND V. G. THOMAS. 2002. Lead-poisoned wildfowl in Spain: a significant threat for human consumers. International Journal of Environmental Health Research 12:301–309.

GUSTAVSSON, P., AND L. GERHARDSSON. 2005. Intoxication from an accidentally ingested lead shot retained in the gastrointestinal tract. Environmental Health Perspectives 113:491–493.

HALDIMANN, M., A. BAUMGARTNER, AND B. ZIMMERLI. 2002. Intake of lead from game meat—a risk to consumers' health? European Food Research and Technology 215:375–379.

HALL, S. L., AND F. M. FISHER. 1985. Lead concentrations in tissues of marsh birds and relationships of feeding habits and grit preference to spent shot ingestion. Bulletin of Environmental Contamination and Toxicology 35:1–8.

HANSPETER, W., AND R. P. KERRY. 2003. Fall diet of Chukars (*Alectoris chukar*) in Eastern Oregon and discovery of ingested lead pellets. Western North American Naturalist 63:402–405.

HENNY, C. J., L. J. BLUS, D. J. HOFFMAN, AND R. A. GROVE. 1994. Lead in hawks, falcons and owls downstream from a mining site on the Coeur d'Alene river, Idaho. Environmental Monitoring and Assessment 29:267–288.

HENNY, C. J., L. J. BLUS, D. J. HOFFMAN, R. A. GROVE, AND J. S. HATFIELD. 1991. Lead accumulation and Osprey production near a mining site on the Coeur d'Alene river, Idaho. Archives of Environmental Contamination and Toxicology 21:415–424.

HERNÁNDEZ, M. 1995. Lead poisoning in a free-ranging Imperial Eagle. Supplement to the Journal of Wildlife Diseases 31, Newsletter.

HERNÁNDEZ, M., AND A. MARGALIDA. 2008 Pesticide abuse in Europe: Effects on the Cinereous Vulture (*Aegypius monachus*) population in Spain. Ecotoxicology 17:264–272.

HIRANO, T., I. KOIKE, AND C. TSUKAHARA. 2004. Lead shots retrieved from the pellets of Eastern Marsh Harriers wintering in Watarase Marsh, Tochigi Prefecture, Japan. Japanese Journal of Ornithology 53:98–100.

HUNT, W. G., W. BURNHAM, C. N. PARISH, K. K. BURNHAM, B. MUTCH, AND J. L. OAKS. 2006. Bullet fragments in deer remains: Implications for lead exposure in avian scavengers. Wildlife Society Bulletin 34:167–170.

IWATA, H., M. WATANABE, E.-Y. KIM, R. GOTOH, G. YASUNAGA, S. TANABE, Y. MASUDA, AND S. FUJITA. 2000. Contamination by chlorinated hydrocarbons and lead in Steller's Sea Eagle and White-tailed Sea Eagle from Hokkaido, Japan. Pages 91–106 *in* M. Ueta and M. J. McGrady (Eds.). First Symposium on Steller's and White-tailed Sea Eagles in East Asia. Wild Bird Society of Japan, Tokyo, Japan.

JACOBSON, E., J. W. CARPENTER, AND M. NOVILLA. 1977. Suspected lead toxicosis in a Bald Eagle. Journal of the American Veterinary Medical Association 171:952–954.

JANSSEN, D. L., J. E. OOSTERHUIS, J. L. ALLEN, M. P. ANDERSON, D. G. KELTS, AND S. N. STANLEY. 1986. Lead poisoning in free-ranging California Condors. Journal of the American

Veterinary Medical Association 189:1115–1117.

JOHANSEN, P., G. ASMUND, AND F. RIGET. 2004. High human exposure to lead through consumption of birds hunted with lead shot. Environmental Pollution 127:125–129.

JOHNSGARD, P. A. 1983. Sandhill Crane. Pages 171–184 *in* Cranes of the World. Croom Helm, London, Canberra.

JONES, J. C. 1939. On the occurrence of lead shot in stomachs of North American gruiformes. Journal of Wildlife Management 3:353–357.

KAISER, G. W., AND K. FRY. 1980. Ingestion of lead shot by Dunlin. The Murrelet 61:37.

KAISER, T. E., W. L. REICHEL, L. N. LOCKE, E. CROMARTIE, A. J. KRYNITSKY, T. G. LAMONT, B. M. MULHERN, R. M. PROUTY, C. J. STAFFORD, AND D. M. SWINEFORD. 1980. Organochlorine pesticide, PCB, and PBB residues and necropsy data for Bald Eagles from 29 states, 1975–77. Pesticide Monitoring Journal 14:145–149.

KALISINSKA, E., W. SALICKI, AND A. JACKOWSKI. 2006. Six trace metals in White-tailed Eagle from northwestern Poland. Polish Journal of Environmental Studies 15:727–737.

KEEL, M. K., W. R. DAVIDSON, G. L. DOSTER, AND L. A. LEWIS. 2002. Northern Bobwhite and lead shot deposition in an upland habitat. Archives of Environmental Contamination and Toxicology 43:318–322.

KENDALL, R. J., AND P. F. SCANLON. 1981. Effects of chronic lead ingestion on reproductive characteristics of Ringed Turtle Doves (*Streptopelia risoria*) and on tissue lead concentrations of adults and their progeny. Environmental Pollution A26:203–213.

KENDALL, R. J., H. P. VEIT, AND P. F. SCANLON. 1981. Histological effects and lead concentrations in tissues of adult male Ringed Turtle Doves (*Streptopelia risoria*) that ingested lead shot. Journal of Toxicology and Environmental Health 8:649–658.

KENNEDY, S., J. P. CRISLER, E. SMITH, AND M. BUSH. 1977. Lead poisoning in Sandhill Cranes. Journal of the American Veterinary Medical Association 171:955–958.

KENNTNER, N., O. KRONE, R. ALTENKAMP, AND F. TATARUCH. 2003. Environmental contaminants in liver and kidney of free-ranging Northern Goshawks (*Accipiter gentilis*) from three regions of Germany. Archives of Environmental Contamination and Toxicology 45:128–135.

KENNTNER, N., G. OEHME, D. HEIDECKE, AND F. TATARUCH. 2004. Retrospektive Untersuchung zur Bleiintoxikation und Exposition mit potenziell toxischen Schwermetallen von Seeadlern (*Haliaeetus albicilla*) in Deutschland. Vogelwelt 125:63–75.

KENNTNER, N., F. TATARUCH, AND O. KRONE. 2001. Heavy metals in soft tissue of White-tailed Eagles found dead or moribund in Germany and Austria from 1993 to 2000. Environmental Toxicology and Chemistry 20: 1831–1837.

KENNTNER, N., F. TATARUCH, AND O. KRONE. 2005. Risk assessment of environmental contaminants in White–tailed Sea Eagles (*Haliaeetus albicilla*) from Germany. Pages 125–127 *in* K. Pohlmeyer (Ed.). Extended Abstracts of the XXVIIth Congress of the International Union of Game Biologists, Hannover 2005. DSV Verlag, Hamburg, Germany.

KENNTNER, N., Y. CRETTENAND, H. J. FÜNFSTÜCK, M. JANOVSKY, AND F. TATARUCH. 2007. Lead poisoning and heavy metal exposure of Golden Eagles (*Aquila chrysaetos*) from the European Alps. Journal of Ornithology 148:173–177.

KEYMER, I. F., AND R. S. STEBBINGS. 1987. Lead poisoning in a Partridge (*Perdix perdix*) after ingestion of gunshot. Veterinary Records 21: 276–277.

KIM, E-Y., R. GOTO, H. IWATA, S. TANABE, Y. MASUDA, AND S. FUJITA. 1999. Preliminary survey of lead poisoning of Steller's Sea Eagle (*Haliaeetus pelagicus*) and White-tailed Eagle (*Haliaeetus albicilla*) in Hokkaido, Japan. Environmental Toxicology and Chemistry 18:448–451.

KNOPPER, L. D., P. MINEAU, A. M. SCHEUHAMMER, D. E. BOND, AND D. T. MCKINNON. 2006. Carcasses of shot Richardson's Ground Squirrels may post lead hazards to scavenging hawks. Journal of Wildlife Management 70:295–299.

KRONE, O., F. WILLE, N. KENNTNER, D. BOERTMANN, AND F. TATARUCH. 2004. Mortality factors, environmental contaminants, and parasites of White-tailed Sea Eagles from Greenland. Avian Diseases 48:417–424.

KRONE, O., T. STIERNBERG, N. KENNTNER, F. TATARUCH, J. KOIVUSAARI, AND I. NUUJA. 2006.

Mortality factors, helminth burden, and contaminant residues in White-tailed Sea Eagles (*Haliaeetus albicilla*) from Finland. Ambio 35:98–104.

KUROSAWA, N. 2000. Lead poisoning in Steller's Sea Eagles and White tailed Sea Eagles. Pages 107–109 *in* M. Ueta and M. J. McGrady (Eds.). First Symposium on Steller's and White-tailed Sea Eagles in East Asia. Wild Bird Society of Japan, Tokyo, Japan.

LANGELIER, K. M., C. E. ANDRESS, T. K. GREY, C. WOOLDRIDGE, R. J. LEWIS, AND R. MARCHETTI. 1991. Lead poisoning in Bald Eagles in British Columbia. Canadian Veterinary Journal 32: 108–109.

LEWIS, J. C., AND E. LEGLER. 1968. Lead shot ingestion by Mourning Doves and incidence in soil. Journal of Wildlife Management 32:476–482.

LEWIS, L. A., R. J. POPPENGA, W. R. DAVIDSON, J. R. FISCHER, AND K. A. MORGAN. 2001. Lead toxicosis and trace element levels in wild birds and mammals at a firearms training facility. Archives of Environmental Contamination and Toxicology 41:208–214.

LOCKE, L. N., AND G. E. BAGLEY. 1967. Lead poisoning in a sample of Maryland Mourning Doves. Journal of Wildlife Management 31: 515–518.

LOCKE, L. N., G. E. BAGLEY, D. N. FRICKIE, AND L. T. YOUNG. 1969. Lead poisoning and Aspergillosis in an Andean Condor. Journal of the American Veterinary Medical Association 155:1052–1056.

LOCKE, L. N., AND M. FRIEND. 1992. Lead poisoning of avian species other than waterfowl. Pages 19–22 *in* D.J. Pain (Ed.). Lead Poisoning in Waterfowl. Proceedings of an IWRB Workshop, Brussels, Belgium, 13–15 June 1991. International Waterfowl and Wetlands Research Bureau Special Publication 16, Slimbridge, UK.

LUMEIJI, J. T., W. T. C. WOLVEKAMP, G. M. BRON-DIETZ, AND A. J. H. SCHOTMAN. 1985. An unusual case of lead poisoning in a Honey Buzzard (*Pernis apivorus*). The Veterinary Quarterly 7:165–168.

MACDONALD, J. W., C. J. RANDALL, H. M. ROSS, G. M. MOON, AND A. D. RUTHVEN. 1983. Lead poisoning in captive birds of prey. Veterinary Record 113:65–66.

MARTIN, P. A., AND G. C. BARRETT. 2001. Exposure of terrestrial raptors to environmental lead: determining sources using stable isotope ratios. Page 84 *in* International Association for Great Lakes Research Conference Program and Abstracts 44, University of Wisconsin—Green Bay, Green Bay, 10–14 June 2001. IAGLR, Ann Arbor, Michigan, USA.

MARTIN, P. A., D. CAMPBELL, AND A. SCHEUHAMMER. 2003. Lead exposure in terrestrial foraging raptors in southern Ontario, 1999–2001. Page 269 *in* International Association for Great Lakes Research Conference Program and Abstracts 46, DePaul University, Chicago, 22–26 June 2003. IAGLR, Ann Arbor, Michigan, USA.

MARTINA, P. A., D. CAMPBELL, K. HUGHES, AND T. MCDANIEL. 2008. Lead in the tissues of terrestrial raptors in southern Ontario, Canada 1951–2001. Science of the Total Environment 391:96–103. Art No. 10.1016/j.scitotenv.2007.11.012\ISSN 0048–9697 2008.

MATEO, R. 1998a. Ingestión de perdigones de plomo en rapaces de las marismas del Guadalquivir. Pages 159–170 *in* La Intoxicación por Ingestión de Perdigones de Plomo en Aves Silvestres: Aspectos Epidemiológicos y Propuestas para su Prevención en España. Doctoral Thesis, Universitat Autònoma de Barcelona, Spain.

MATEO, R. 1998b. La intoxicación por ingestión de objetos de plomo en aves: una revisión de los aspectos epidemiológicos y clínicos. Pages 5–44 *in* La Intoxicación por Ingestión de Perdigones de Plomo en Aves Silvestres: Aspectos Epidemiológicos y Propuestas para su Prevención en España. Doctoral Thesis, Universitat Autònoma de Barcelona, Spain.

MATEO, R., R. CADENAS, M. MANEZ, AND R. GUITART. 2001. Lead shot ingestion in two raptor species from Doñana, Spain. Ecotoxicology and Environmental Safety 48:6–10.

MATEO, R., J. ESTRADA, J-Y. PAQUET, X. RIERA, L. DOMÍNGUEZ, R. GUITART, AND A. MARTÍNEZ-VILALTA. 1999. Lead shot ingestion by Marsh Harriers (*Circus aeruginosus*) from the Ebro Delta, Spain. Environmental Pollution 104:435–440.

MATEO, R., R. MOLINA, J. GRÍFOLS, AND R. GUITART. 1997. Lead poisoning in a free-

ranging Griffon Vulture (*Gyps fulvus*). The Veterinary Record 140: 47–48.

MATEO, R., M. TAGGART, AND A. A. MEHARG. 2003. Lead and arsenic in bones of birds of prey from Spain. Environmental Pollution 126:107–114.

MATEO, R., M. RODRIGUEZ-DE LA CRUZ, D. VIDAL, M. REGLERO, AND P. CAMARERO. 2007. Transfer of lead from shot pellets to game meat during cooking. Science of the Total Environment 372:480–485.

MATSUDA, H. 2003. Challenges posed by the precautionary principle and accountability in ecological risk assessment. Environmetrics 14: 245–254.

MCQUIRTER, J. L., S. J. ROTHENBERG, G. A. DINKINS, V. KONDRASHOV, M. MANOLO, AND A. C. TODD. 2004. Change in blood lead concentration up to 1 year after a gunshot wound with a retained bullet. American Journal of Epidemiology 159:683–692.

MEHARG, A. A., D. J. PAIN, R. M. ELLAM, R. BAOS, V. OLIVE, A. JOYSON, N. POWELL, A. J. GREEN, AND F. HIRALDO. 2002. Isotopic identification of the sources of lead contamination for White Storks (*Ciconia ciconia*) in a marshland ecosystem (Doñana, S.W. Spain). Science of the Total Environment 300: 81–86.

MERETSKY, V. J., N. F. R. SNYDER, S. R. BEISSINGER, D. A. CLENDENEN, AND J. W. WILEY. 2000. Demography of the California Condor: implications for reestablishment. Conservation Biology 14:957–967.

MIGLIORINI, M., G. PIGINO, N. BIANCHI, F. BERNINI, AND C. LEONZIO. 2004. The effects of heavy metal contamination on the soil arthropod community of a shooting range. Environmental Pollution 129:331–340.

MILLER, M. J. R., M. E. WAYLAND, E. H. DZUS, AND G. BORTOLOTTI. 2000. Availability and ingestion of lead shotshell pellets by migrant Bald Eagles in Saskatchewan. Journal of Raptor Research 34:167–174.

MILLER, M. J. R., M. E. WAYLAND, E. H. DZUS, AND G. BORTOLOTTI. 2001. Exposure of migrant Bald Eagles to lead in prairie Canada. Environmental Pollution 112:153–162.

MUDGE, G. P. 1992. Options for alleviating lead poisoning: a review and assessment of alternatives to the use of non-toxic shot. Pages 23–25 *in* D.J. Pain (Ed.). Lead Poisoning in Waterfowl. Proceedings of an IWRB Workshop, Brussels, Belgium, 13–15 June 1991. International Waterfowl and Wetlands Research Bureau Special Publication 16, Slimbridge, UK.

NATIONAL WILDLIFE HEALTH LABORATORY (NWHL). 1985. Lead poisoning in non-waterfowl avian species. United States Fish and Wildlife Service Unpublished Report.

NOER, H., AND J. MADSEN. 1996. Shotgun pellet loads and infliction rates in Pink-footed Geese *Anser brachyrhynchus.* Wildlife Biology 2:65–82.

OAKS, J. L., M. GILBERT, M. Z. VIRANI, R. T. WATSON, C. U. METEYER, B. A. RIDEOUT, H. L. SHIVAPRASAD, S. AHMED, M. J. I. CHAUDHRY, M. ARSHAD, S. MAHMOOD, A. ALI, AND A. A. KHAN. 2004. Diclofenac residues as the cause of vulture decline in Pakistan. Nature 2317 Letters: 1–4.

PAIN, D. J. 1990a. Lead shot ingestion by waterbirds in the Camargue, France: an investigation of levels and interspecific differences. Environmental Pollution 66:273–285.

PAIN, D. J. 1990b. Lead poisoning of waterfowl: a review. Pages 172–181 *in* G. Matthews (Ed.). Managing Waterfowl Populations. The International Waterfowl and Wetlands Research Bureau, Slimbridge, UK.

PAIN, D. J. 1991a. Lead poisoning in birds: an international perspective. Acta XX Congressus Internationalis Ornithologici, 2343–2352.

PAIN, D. J. 1991b. Why are lead-poisoned waterfowl rarely seen? The disappearance of waterfowl carcasses in the Camargue, France. Wildfowl 42:118–122.

PAIN, D. J. 1992. Lead poisoning in waterfowl: a review. Pages 7–13 *in* D.J. Pain (Ed.). Lead Poisoning in Waterfowl. Proceedings of an IWRB Workshop, Brussels, Belgium, 13–15 June 1991. International Waterfowl and Wetlands Research Bureau Special Publication 16, Slimbridge, UK.

PAIN, D. J. 1996. Lead in waterfowl. Pages 251–262 *in* W. M. Beyer, G. H. Heinz, and A. W. Redman-Norwood (Eds.). Environmental Con–taminants in Wildlife: Interpreting Tissue Concentrations. Lewis Publishers, Boca Raton, Florida, USA.

PAIN, D. J., AND C. AMIARD-TRIQUET. 1993. Lead poisoning in raptors in France and elsewhere. Ecotoxicology and Environmental Safety 25: 183–192.

PAIN, D. J., C. AMIARD-TRIQUET, C. BAVOUX, G. BURNELEAU, L. EON, AND P. NICOLAU-GUILLAUMET. 1993. Lead poisoning in wild populations of Marsh Harrier (*Circus aeruginosus*) in the Camargue and Charente-Maritime, France. Ibis 135:379–386.

PAIN, D. J., C. BAVOUX, AND G. BURNELEAU. 1997. Seasonal blood lead concentrations in Marsh Harriers (*Circus aeruginosus*) from Charente-Maritime, France: relationship with hunting season. Biological Conservation 81:1–7.

PAIN, D. J., A. A. MEHARG, M. FERRER, M. TAGGART, AND V. PENTARIANI. 2004. Lead concentrations in bones and feathers of the globally threatened Spanish Imperial Eagle. Biological Conservation 121:603–610.

PAIN, D. J., J. SEARS, AND I. NEWTON. 1995. Lead concentrations in birds of prey in Britain. Environmental Pollution 87:173–180.

PAIN, D. J., I. CARTER, A. W. SAINSBURY, R. F. SHORE, P. EDEN, M. A. TAGGART, S. KONSTANTINOS, L. A. WALKER, A. A. MEHARG, AND A. RAAB. 2007. Lead contamination and associated disease in captive and reintroduced Red Kites *Milvus milvus* in England. Science of the Total Environment 376:116–127.

PATTEE, O. H., P. H. BLOOM, J. M. SCOTT, AND M. R. SMITH. 1990. Lead hazards within the range of the California Condor. The Condor 92: 931–937.

PATTEE, O. H., AND S. K. HENNES. 1983. Bald Eagles and waterfowl: the lead shot connection. Pages 230–237 *in* 48th North American Wildlife Conference 1983. The Wildlife Management Institute, Washington, DC, USA.

PATTEE, O. H., S. N. WIEMEYER, B. M. MULHERN, L. SILEO, AND J. W. CARPENTER. 1981. Experimental lead-shot poisoning in Bald Eagles. Journal of Wildlife Management 45:806–810.

PLATT, J. B. 1976. Bald Eagles wintering in the Utah desert. American Birds 30: 783–788.

POTTS, G. R. 2005. Incidence of ingested lead gunshot in wild Grey Partridges. European Journal of Wildlife Research 51:31–34.

QUORTRUP, E. R., AND J. E. SHILLINGER. 1941. 3,000 wild bird autopsies on western lake areas. Journal of the American Veterinary Medical Association 99:382–397.

REDIG, P. T., E. M. LAWLER, S. SCHWARTZ, J. L. DUNNETTE, B. STEPHENSON, AND G. E. DUKE. 1991. Effects of chronic exposure to sub-lethal concentrations of lead acetate on heme synthesis and immune function in Red-tailed Hawks. Archives of Environmental Contamination and Toxicology 21:72–77.

REDIG, P. T., C. M. STOWE, D. M. BARNES, AND T. D. ARENT. 1980. Lead toxicosis in raptors. Journal of the American Veterinary Association 177:941–943.

REICHEL, W. L., S. K. SCHMELING, E. CROMARTIE, T. E. KAISER, A. J. KRYNITSSKY, T. G. LAMONT, B. M. MULHERN, R. M. PROUTY, D. J. STAFFORD, AND D. M. SWINEFORD. 1984. Pesticide, PCB and lead residues and necropsy data from Bald Eagles for 32 states—1978–81. Environmental Monitoring and Assessment 4:395–403.

REISER, M. H., AND S. A. TEMPLE. 1981. Effect of chronic lead ingestion on birds of prey. Pages 21–25 *in* J. E. Cooper and A. G. Greenwood (Eds.). Recent Advances in the Study of Raptor Diseases. Chiron Publications, Keighley, UK.

RODRIGUE, J., R. MCNICOLL, D. LECLAIR, AND J. F. DUCHESNE. 2005. Lead concentrations in Ruffed Grouse, Rock Ptarmigan, and Willow Ptarmigan in Quebec. Archives of Environ–mental Contamination and Toxicology 49:97–104.

RODRIGUEZ-RAMOS, J., V. GUTIERREZ, U. HÖFLE, R. MATEO, L. MONSALVE, E. CRESPO, J. M. BLANCO. 2009. Lead in Griffon and Cinereous Vultures in Central Spain: Correlations between clinical signs and blood lead levels. Extended abstract *in* R. T. Watson, M. Fuller, M. Pokras, and W. G. Hunt (Eds.). Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans. The Peregrine Fund, Boise, Idaho, USA. DOI 10.4080/ilsa.2009.0213

SAINSBURY, A. W., P. M. BENNETT, AND J. K. KIRKWOOD. 1995. The welfare of free-living wild animals in Europe: harm caused by human activities. Animal Welfare 4:183–206.

SANDERSON, G. C., AND F. C. BELLROSE. 1986. A review of the problem of lead poisoning in waterfowl. Illinois Natural History Survey, Special Publication 4:1–34.

SCHEUHAMMER, A. M. 1987. The chronic toxicity of aluminium, cadmium, mercury, and lead in birds: a review. Environmental Pollution 46: 263–295.

SCHEUHAMMER, A. M., D. E. BOND, N. M. BURGESS, AND J. RODRIGUE. 2003. Lead and stable isotope ratios in soil, earthworms and bones of American Woodcock (*Scolopax minor*) from eastern Canada. Environmental Toxicology and Chemistry 22:2585–2591.

SCHEUHAMMER, A. M., AND S. L. NORRIS. 1995. A review of the environmental impacts of lead shotshell ammunition and lead fishing weights in Canada. Canadian Wildlife Service, Occasional Paper 88.

SCHEUHAMMER, A. M., AND S. L. NORRIS. 1996. The ecotoxicology of lead shot and lead fishing weights. Ecotoxicology 5:279–295.

SCHEUHAMMER, A. M., J. A. PERRAULT, E. ROUTHIER, B. M. BRAUNE, AND G. D. CAMPBELL. 1998. Elevated lead concentrations in edible portions of game birds harvested with lead shot. Environmental Pollution 102:251–257.

SCHEUHAMMER, A. M., C. A. ROGERS, AND D. BOND. 1999. Elevated lead exposure in American Woodcock (*Scolopax minor*) in eastern Canada. Archives of Environmental Contami–nation and Toxicology 36:334–340.

SCHEUHAMMER, A. M., AND D. M. TEMPLETON. 1998. Use of stable isotope ratios to distinguish sources of lead exposure in birds. Ecotoxicology 7:37–42.

SCHULZ, J. H., J. J. MILLSPAUGH, B. E. WASHBURN, G. R. WESTER, J. T. LANIGAN, AND J. C. FRANSON. 2002. Spent-shot availability and ingestion on areas managed for Mourning Doves. Wildlife Society Bulletin 30:112–120.

SCHULZ, J. H., P. I. PADDING, AND J. J. MILLSPAUGH. 2006. Will Mourning Dove crippling rates increase with nontoxic-shot regulations? Wildlife Society Bulletin 34:861–865.

SERGEYEV, A. A., AND N. A. SHULYATIEVA. 2005. Meat quality of birds after using lead shot. Pages 472–473 *in* K. Pohlmeyer (Ed.). Ex–tended Abstracts of the XXVIIth Congress of the International Union of Game Biologists, Hannover 2005. DSV Verlag, Hamburg, Germany.

SHARLEY, A. J., L. W. BEST, J. LANE, AND P. WHITEHEAD. 1992. An overview of lead poisoning in Australian waterfowl and implications for management. Pages 73–77 *in* D.J. Pain (Ed.). Lead Poisoning in Waterfowl. Proceedings of an IWRB Workshop, Brussels, Belgium, 13–15 June 1991. International Waterfowl and Wetlands Research Bureau Special Publication 16, Slimbridge, UK.

SILEO, L., L. H. CREEKMORE, D. J. AUDET, M. R. SNYDER, C. U. METEYER, J. C. FRANSON, L. N. LOCKE, M. R. SMITH, AND D. L. FINLEY. 2001. Lead poisoning of waterfowl by contaminated sediment in the Coeur d'Alene River. Archives of Environmental Contamination and Toxicology, 41:364–368.

SILEO, L., AND S. I. FEFER. 1987. Paint chip poisoning of Laysan Albatross at Midway atoll. Journal of Wildlife Diseases 23:432–437.

SNYDER, N. F., AND H. A. SNYDER. 2000. The California Condor. Academic Press, New York, USA.

SNYDER, N. F., H. A. SNYDER, J. L. LINCER, AND R. T. REYNOLDS. 1973. Organochlorines, heavy metals and the biology of North American accipiters. Biosciences 23:300–305.

SORVARI, J., R. ANTIKAINEN, AND O. PYY. 2006. Environmental contamination at Finnish shooting ranges—the scope of the problem and management options. Science of the Total Environment 366:21–31.

STEHLE, V. S. 1980. Orale Bleivergiftung bei Greifvögeln (Falconiformes)—Vorläufige Mitteilung. Kleintier Prakis 25:309–310.

STENDELL, R. C., R. I. SMITH, K. P. BURNHAM, AND R. E. CHRISTENSEN. 1979. Exposure of waterfowl to lead: a nationwide survey of residues in wing bones of seven species, 1972–1973. U.S. Fish and Wildlife Service Special Scientific Report Wildlife 223:1–12.

STENDELL, R. C., J. W. ARTMANN, AND E. MARTIN. 1980. Lead residues in Sora Rails from Maryland. Journal of Wildlife Management 44:525–527.

STEVENSON, A. L., A. M. SCHEUHAMMER, AND H. M. CHAN. 2005. Effects of nontoxic shot regulations on lead accumulation in ducks and American Woodcock in Canada. Archives of Environmental Contamination and Toxicology 48:405–413.

STODDARD, H. L. 1931. The Bobwhite Quail, its habits, preservation and increase. Charles Scribner's Sons, New York, USA.

STONE, W. B., AND S. A. BUTKAS. 1978. Lead poisoning in a wild turkey. New York Fish and Game Journal 25:169.

SZYMCZAK, M. R. 1978. Steel shot use on a goose hunting area in Canada. Wildlife Society Bulletin 6:217–225.

TAVERNIER, P., S. ROELS, K. BAERT, K. HERMANS, F. PASMANS, AND K. CHIERS. 2004. Lead intoxication by ingestion of lead shot in racing pigeons (*Columba livia*). Vlaams Diergeneeskundig Tijdschrift 73:307–309.

THOMAS, V. G. 1997. The environmental and ethical implications of lead shot contamination of rural lands in north America. Journal of Agricultural and Environmental Ethics 10:41–54.

THOMAS, V. G., AND R. GUITART. 2005. Role of international conventions in promoting avian conservation through reduced lead toxicosis: progression towards a non-toxic agenda. Bird Conservation International 15:147–160.

TSUJI, L. J. S., E. NIEBOER, J. D. KARAGATZIDES, R. M. HANNING, AND B. KATAPATUK. 1999. Lead shot contamination in edible portions of game birds and its dietary implications. Ecosystem Health 5:183–192. Art No. ISSN 1076-2825.

UETA, M., AND V. MASTEROV. 2000. Estimation by a computer simulation of population trend of Steller's Sea Eagles. Pages 111–116 *in* M. Ueta and M. J. McGrady (Eds.). First Symposium on Steller's and White-tailed Sea Eagles in East Asia. Wild Bird Society of Japan, Tokyo, Japan.

VEIGA, J. 1985. Contribution à l'étude du regime alimentaire de la Bécassine Sourde (*Lymnocryptes minimus*). Gibier Faune Sauvage 1:75–84.

VEIT, H. P., R. J. KENDALL, AND P. F. SCANLON. 1982. The effect of lead shot ingestion on the testes of adult Ringed Turtle Doves (*Streptophelia risoria*). Avian Diseases 27:442–452.

VON KUSTER, D. D., AND D. SCHNEBERGER. 1992. Diet of the Great Horned Owl in central Saskatchewan. Blue Jay 50: 195–200.

VYAS, N. B., J. W. SPANN, G. H. HEINZ, W. N. BEYER, J. A. JAQUETTE, AND J. M. MENGEL–KOCH. 2000. Lead poisoning of passerines at a trap and skeet range. Environmental Pollution 107:159–166.

WAYLAND, M., AND T. BOLLINGER. 1999. Lead exposure and poisoning in Bald Eagles and Golden Eagles in the Canadian prairie provinces. Environmental Pollution 104:341–350.

WAYLAND, M., L. K. WILSON, J. E. ELLIOTT, J. E. MILLER, T. BOLLINGER, M. MCADIE, K. LANGELIER, J. KEATING, AND J. M. W. FROESE. 2003. Mortality, morbidity and lead poisoning of eagles in western Canada 1986–1998. Journal of Raptor Research 37:8–18.

WIEMEYER, S. N., R. M. JUREK, AND J. F. MOORE. 1986. Environmental contaminants in surrogates, foods, and feathers of California Condors (*Gymnogyps californianus*). Environmental Monitoring and Assessment 6:91–111.

WIEMEYER, S. N., A. J. KRYNITSKY, AND S. R. WILBUR. 1983. Environmental contaminants in tissues, foods, and feces of California Condors. Pages 4727–4739 *in* S. R. Wilbur and J. A. Jackson, (Eds.). Vulture Biology and Management. University of California Press, Los Angeles, California, USA.

WIEMEYER, S. N., J. M. SCOTT, M. P. ANDERSON, P. H. BLOOM, AND C. J. STAFFORD. 1988. Environmental contaminants in California Condors. Journal of Wildlife Management 52:238–247.

WINDINGSTAD, R. M., S. M. KERR, L. N. LOCKE, AND J. J. HUNT. 1984. Lead poisoning of Sandhill Cranes (*Grus canadensis*). Prairie Naturalist 16:21–24.

WOBESTER, G., AND A. G. WOBESTER. 1992. Carcass disappearance and estimation of mortality in a simulated die-off of small birds. Journal of Wildlife Diseases 28:548–554.